Yellow Fever

HYGIENE & DISEASES
OF
WARM CLIMATES.

EDITED BY

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EDINBURGH AND LONDON:
YOUNG J. PENTLAND.
1893.

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CHAPTER VII.

YELLOW FEVER.

BY GEORGE M. STERNBERG, M.D., U.S.A.

Definition.—Yellow fever is a specific infectious disease which is characterised by a single febrile paroxysm of short duration (two to five days), followed by a period of great depression of the vital powers (so-called stage of calm), by the presence of albumen in the urine, by an icteric colour of the skin, and by a tendency to passive hæmorrhages from mucous surfaces—especially from the stomach—producing " black vomit."

ETIOLOGY.

A Miasmatic Contagious Disease.—Yellow fever is contracted by exposure in infected localities, and not directly by contact with the sick. This fact is established by abundant observations, although some physicians still look upon it as a strictly contagious disease. No doubt, as in cholera and in typhoid fever, a specific infectious agent is given off from the bodies of the sick, which when it finds a favourable nidus may establish a new centre of infection; and, as in the diseases named, it seems probable that this specific infectious agent is contained in the alvine discharges of the sick. That it is not given off from the general surface of the body, we infer from the well-established fact that the disease is not contracted by those intimately associated with the sick as a result of such association. During the epidemic prevalence of yellow fever, many physicians and nurses may fall victims to the disease, as they are necessarily exposed in infected localities. But when persons successively fall sick in the same house, or on board ship, they are not infected one from another by direct personal contact, but contract the disease from a common source—the infected locality.

It is well known to the people of the city of Mexico that a visit to the sea-coast city of Vera Cruz during the epidemic season is

likely to result in an attack of yellow fever. It is also well established that those who fall sick with the disease after their return to the city of Mexico never communicate it to others who are closely associated with them as attendants, etc. The same is true at the health-resort, Petropolis, located in the mountains within a few hours' ride of the city of Rio Janeiro. Frequently individuals fall sick in Petropolis who have visited the infected city of Rio. Never do they communicate the disease to others. This is also the experience of the physicians in charge of hospitals—*e.g.* the Charity Hospital of New Orleans; so long as the hospital and its vicinity remain uninfected, cases do not originate in the hospital, although yellow fever patients may be treated in the wards along with unacclimatised persons suffering from other diseases, and be cared for by susceptible attendants.

In his report upon the camps established near Memphis in the epidemics of 1878 and 1879, Colonel Cameron makes the following statement: "It was found necessary that the officer in authority should set an example of constant indifference to attack in order to appease, as far as possible, the constant anxiety of the population under his charge. Especially was this true in 1878, as depopulation went on slowly that year, and infected people poured daily into the camps from the more pestilential portions of the city. Very many reached camp with the fever on them, so that as many as seventeen persons fell victims in one night, not a few in their tents. In no instance, however, did they communicate the disease to their families or bed-fellows, as far as could be traced."

In the same epidemic (1878), Dr. Minor reports that over thirty cases were discovered among refugees in Cincinnati, O., and says: "No physician or nurse contracted the disease, and in no instance did it exhibit any tendency to spread." The same was true in Nashville the same year; twenty imported cases occurred in different parts of the city without any local cases resulting from them.[1] Evidence of this kind could be extended to fill a volume; but sufficient has been presented to establish the statement made, and the reader may be referred to the "proofs of non-contagion" in the second volume of the classical work of La Roche (pp. 236–566).

Epidemic Development.—The development of an epidemic of yellow fever, in places removed from the foci of the disease, depends upon—(*a*) the introduction of the specific cause by yellow fever patients, or through infected articles—fomites; (*b*) local conditions which favour the multiplication of the specific germ external to the body; (*c*) favourable meteorological conditions; (*d*) the presence of susceptible individuals in the infected locality.

Usually new infected centres are established by the arrival of individuals who fall sick of the disease as a result of exposure in previously infected localities.

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[1] *Report of Nashville Board of Health.*

That infected centres may be established independently of the arrival of sick persons is, however, beyond question.

A striking instance of this is afforded by an outbreak which occurred at Madrid in 1878. A circumscribed epidemic was developed in this city about the 1st of September, which resulted in a mortality of thirty-five out of fifty cases taken sick. This outbreak was traced to importation, although all of the cases occurred among the permanent residents of the infected area. Associated with the young people who first fell sick, crowded in the same rooms with them to the number of ten or fifteen in a room, were a number of soldiers recently returned from Cuba with their baggage. These men had themselves suffered from yellow fever in Cuba, or were acclimatised by long residence there.

We have on record instances which appear to be authentic of the development of an epidemic as the result of the opening of a trunk containing infected clothing, sent from a locality where the disease was prevailing to one previously healthy. Epidemics have also been traced to the unloading of earth ballast from the shores of an infected port upon the wharves of a healthy place in the yellow fever zone.

The first cases of local origin in an epidemic do not, as a rule, occur until some time has elapsed after the arrival of the infected ship, or fomites, or sick person responsible for the introduction of the "germ." The interval may vary from a few days to several weeks, according as local conditions are favourable or otherwise for the development of the infectious agent.

In the great epidemic of 1878, which was traced by Dr. Choppin, President of the Louisiana State Board of Health, to importation by the steamship *Emily B. Souder*, which arrived from Havana on the 23rd of May, the first cases of local origin did not occur until after an interval of five or six weeks. But these first cases occurred, according to Dr. Choppin, in the immediate vicinity of the houses in which two of the officers of the *Souder* (Clarke the purser, and Elliott, one of the engineers) died soon after the arrival of that vessel.

Altitude.—The recorded facts do not justify the conclusion that the limitations as to altitude depend solely upon the lower temperature of elevated regions. As pointed out by Hirsch, "the disease stops short at many points in the West Indies where the climate is still in the highest degree tropical. On the other hand, there have been epidemics in cool weather at very considerable altitude, as, for example, at Newcastle, in Jamaica" (elevation about 4000 feet). In the Antilles the disease has rarely appeared at a height of more than 700 feet. In Mexico it has prevailed, however, at Cordova (2500), but is unknown in the cities of Orizaba, Jalapa, and Puebla, which have elevations of more than 3000 feet. In

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Spain a single limited outbreak has occurred at Madrid, which is about 2000 feet above the sea-level; but with this exception the altitudinal range has rarely exceeded 1000 feet. In the United States the most elevated locality in which the disease has prevailed as an epidemic is Chattanooga, Tenn., which is 745 feet above the sea level.

Yellow fever is essentially a disease of the sea coast; and while in great epidemics it may become widely diffused in the interior, it follows, for the most part, the course of navigable rivers.

It is a disease of cities and towns of considerable size, and rarely extends to small country villages, or spreads among the scattered rural population.

In seaport towns it frequently makes its first appearance in the vicinity of the wharves, or in localities frequented by sailors.

Sanitary Conditions.—Above all, yellow fever is a disease which is influenced by local unsanitary conditions. In those places where it is endemic, it haunts the low-lying and filthy portions of the town; and in epidemics it exhibits a marked preference for towns which are in an unsanitary condition. It frequently happens when a town is invaded that the disease is limited, for a considerable time at least, to the filthy districts of the place, in which the degraded victims of poverty and vice congregate in ill ventilated apartments, surrounded by the filth which accumulates in such localities when not kept under a rigid sanitary supervision.

Decomposing organic matter of *animal* origin seems to furnish an especially favourable nidus for the germ. This is shown by its favourite haunts, and by the fact that in marshy places, in the vicinity of cities where it prevails, and where *vegetable* decomposition is active, it does not effect a lodgment. The influence of putrefying organic matter of animal origin in the causation of epidemics has several times been made apparent. Parkes, the famous English hygienist, maintained the fæcal origin of the disease; and there seems to be good reason for the belief that the accumulation of this kind of filth in exposed situations is favourable to the development of an epidemic.

Meteorological Conditions.—*Temperature.*—These control in a most decided manner the prevalence of the disease in localities where it is endemic, and its epidemic extension when new infected centres are established among a susceptible population. The influence of temperature is shown by the fact that it is a disease of the tropics and of hot seasons; that it prevails throughout the year in the cities of Rio Janeiro, Havana, and Vera Cruz, although to a much less extent during the cool season; while in

more temperate regions its prevalence is limited to the summer season. It does not prevail as an endemic disease in places which have a mean winter temperature much below 65° F.; and, as a rule, epidemics are not developed at a lower temperature than 75° to 80° F. The approach of cool weather checks the progress of an epidemic, and it is arrested completely when the temperature falls to the freezing point. There are, however, numerous facts which indicate that the infectious agent, although rendered inactive, is not destroyed by a freezing temperature. Epidemics which have been checked by frost have been revived by the recurrence of warm weather; and in certain instances in temperate regions the germ has survived the winter, and a second epidemic has occurred without a new importation (Memphis, 1878–79; Cadiz, 1800–1; Malaga, 1808–9). Epidemics which originate early in the season often terminate before there is frost, simply because the susceptible material is exhausted; but when strangers venture within the infected area they furnish evidence of the continued activity of the morbific poison, by falling victims to the disease.

The influence of season is shown by the following table:—

MORTALITY FROM YELLOW FEVER IN THE CITY OF HAVANA FOR TEN YEARS—1870 TO 1879 INCLUSIVE.[1]

Month.	1870.	1871.	1872.	1873.	1874.	1875.	1876.	1877.	1878.	1879.	Average of Ten Years.
January, .	6	18	20	32	7	16	24	8	26	11	17
February, .	4	23	13	23	4	16	24	9	13	13	14
March, .	4	12	4	27	18	32	29	11	5	6	15
April, .	6	54	4	37	22	34	33	8	28	13	24
May,. .	14	91	13	127	85	32	103	16	53	40	57
June, .	66	201	68	378	172	142	292	143	184	237	188
July, .	112	234	68	416	361	187	675	249	504	475	328
August, .	201	138	70	127	416	144	250	285	374	417	242
September,	91	72	59	35	186	102	97	234	179	148	120
October, .	77	55	38	28	91	109	42	185	106	44	78
November,	49	51	85	5	42	105	31	150	53	31	60
December,	35	42	73	9	21	82	19	76	34	9	40
Total, .	665	991	515	1244	1425	1001	1619	1374	1559	1444	1183

Although the development of an epidemic seems to require a comparatively high temperature (75° to 80° F.), experience shows that it may continue in full force at a much lower temperature (60° to 70° F.) when local conditions are favourable and a susceptible population is exposed in the infected area.

Atmospheric Humidity and Rainfall.—Another factor of import-

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[1] Chaillé, *Report to National Board of Health.*

ance in the etiology of yellow fever epidemics is atmospheric moisture and precipitation. Yellow fever is a disease of the sea coast and of the margins of great rivers, and it does not prevail in arid, desert places in the interior, although the elevation may be but a little above the sea level, and the temperature extremely high. There is reason to think that this difference is largely due to the difference as to moisture in the atmosphere and in the soil. Some authors have insisted especially upon the presence of moisture in the atmosphere, almost to the point of saturation, as an essential condition for the development of an epidemic. But, on the other hand, epidemics have sometimes occurred in unusually dry seasons, as at New Orleans in 1841. In Martinique, according to Dutrouleau, yellow fever has sometimes committed its greatest ravages after and during the driest season.

Heavy rains, by purifying the air and cleansing the streets and sewers of an infected city, exercise a favourable effect upon its sanitary condition, and in the tropics the commencement of the rainy season often puts an end to the prevailing epidemic. It is probable that the statement made by some authors, that upon the coast of Guiana, and elsewhere in the tropics, dry weather is favourable to the spread of the disease, is but another way of stating the fact that the heavy rains of tropical regions are unfavourable, the dry weather being only dry by comparison.

Wind.—There is no evidence that the infectious agent of yellow fever can be conveyed by the wind, in an active condition, to any considerable distance; and the wind has but little to do with the dissemination of the disease. In cities, the extension of an epidemic from centres, resulting from the importation of cases, or fomites, is usually quite gradual, and independent of the prevailing currents of air.

In the comparatively small and land-locked harbour of Havana, vessels which anchor some distance from the shore, and which are kept in a good sanitary condition, do not suffer from yellow fever unless unacclimatised members of the crew are permitted to go on shore. Vessels lying at the wharves, on the contrary, are very likely to become infected.

Bacteriology.—The facts heretofore stated lead to the inference that the specific infectious agent in yellow fever is a living micro-organism which, under favourable meteorological conditions, multiplies external to the human body in unsanitary localities, where it finds a suitable pabulum for its growth.

But the correctness of this inference has not as yet been established by an experimental demonstration, and we are still

ignorant of the morphological and biological characters of the "yellow fever germ," notwithstanding the claims which have been made by several investigators to have discovered it. This assertion is based upon the personal investigations of the writer, made in Brazil, in Cuba, in Mexico, and in the United States, in accordance with an Act of Congress (1887) authorising such an investigation. The results of these investigations are given in detail in my *Report on the Etiology and Prevention of Yellow Fever* (Government Printing Office, Washington, 1890).

Briefly stated, these results were as follows:—

Forty-two autopsies were made in typical cases of yellow fever, and seventeen autopsies in other diseases for comparative researches.

Aërobic and anaërobic cultures were made from the blood, the liver, the kidney, the urine, the stomach, and the intestine.

The experimental data recorded show that—

The most approved bacteriological methods fail to demonstrate the constant presence of any micro-organism in the blood and tissues of yellow fever cadavers peculiar to the disease.

The micro-organisms which are sometimes obtained in cultures from the blood and tissues are present in comparatively small numbers; and the one most frequently found (*Bacterium coli commune*) is present in the intestine of healthy individuals, and consequently its occasional presence cannot have any etiological import.

A few scattered bacilli are present in the liver, and probably in other organs, at the moment of death. This is shown by preserving portions of the liver, obtained at a recent autopsy, in an antiseptic wrapping.

At the end of twenty-four to forty-eight hours the interior of a piece of liver so preserved contains a large number of bacilli of various species, the most abundant being those heretofore mentioned as occasionally found in fresh liver tissue, viz. *Bacterium coli commune* and *Bacillus cadaverus*.

Blood, urine, and crushed liver tissue, obtained from a recent autopsy, are not pathogenic in moderate amounts for rabbits or guinea-pigs.

Liver tissue preserved in an antiseptic wrapping at a temperature of 28° to 30° C., for forty-eight hours, is very pathogenic for guinea-pigs when injected subcutaneously.

This pathogenic power appears to be due to the micro-organisms present, and to the toxic products developed as a result of their growth. It is not peculiar to yellow fever, inasmuch as material preserved in the same way at comparative autopsies, in which death resulted from accident or other diseases, has given a similar result.

Having failed to demonstrate the presence of a specific "germ" in the blood and tissues, it seems probable that it is to be found in

the alimentary canal, as is the case in cholera. But the extended researches made, and recorded in my report, show that the contents of the intestines of yellow fever cases contain a great variety of bacilli, and not a nearly pure culture of a single species, as is the case in recent and typical cases of cholera.

Comparatively few liquefying bacilli are found in the fæces discharged during life, or in the intestinal contents collected soon after death from yellow fever cadavers. On the other hand, non-liquefying bacilli are very abundant.

The one most constantly and abundantly present is the *Bacterium coli commune* of Escherich.

This is associated with various other bacilli, some of which are strict anaërobics and some facultative anaërobics.

Most of these bacilli have been excluded by the fact that they are not constantly found, or that they have been obtained from the contents of the intestine of healthy persons, or of those dying from other diseases than yellow fever.

Some of the micro-organisms present in the dejecta of yellow fever patients, as shown by stained smear-preparations, have not developed in the cultures made, either aërobic or anaërobic. One extremely slender filiform bacillus, which can only be seen with high powers, and which is quite abundant in some of my preparations, has never been obtained in the cultures made; and no doubt there are others in the same category.

That the yellow fever germ is strictly anaërobic, or that it will only grow in a special nidus, may be inferred from certain facts relating to the extension of epidemics.

Media of Propagation.—There is no evidence that yellow fever is propagated by contamination of the supply of drinking water, as frequently, and probably usually, occurs in the case of typhoid fever and cholera. Moreover, epidemics extend in a more deliberate manner, and are restricted within a more definite area, than is the case with cholera and typhoid fever. It is usually at least ten days or two weeks after the arrival of an infected vessel, or of a person sick with the disease, before cases of local origin occur; and these cases occur in the immediate vicinity of the imported case or of the infected vessel. When the disease has effected a lodgment, the area of infection extends slowly, and usually has well-defined boundaries. In towns and cities having a common water supply one district remains perfectly healthy, while another, and usually the most filthy one, may be decimated by the scourge.

Micro-Organisms which have been described as the Specific Cause of Yellow Fever.—*Cryptococcus xanthogenicus*

of Dr. Domingos Freire, of Brazil.—In his principal work, published in 1885, Freire gives the following account of his cryptococcus:—

"When we follow with care and attention the march of the development which characterises the germs which produce yellow fever, we acquire a certainty that, commencing to present themselves under the form of little points almost imperceptible, they afterwards gradually increase in diameter until they attain considerable dimensions; so that the little beings, which at the outset had the appearance of little grains of sand, not measuring more than 0·001 millimetre to 0·002 millimetre in diameter, arrive little by little to such a development that they reach the dimensions of 0·005, 0·007, 0·008 millimetre, and sometimes even more in certain conditions. When they have attained the adult age, these cells are broken at divers points, and discharge their contents, composed of spores already formed, mixed with a viscous substance of a yellow colour, composed of a pigment and a protoplasmic substance, and of the liquids elaborated by the cells."

In an address delivered in Paris in 1887, Freire repeats this account of the mode of development of his cryptococcus. He says:—

"Each adult cell is ruptured at one or several points, and allows to escape its contents, composed of germs which are to perpetuate the species, and two pigments—one yellow, destined to infiltrate the tissues, and to produce the icteric colour which has given name to the malady; the other black, insoluble, and destined to be carried along the circulatory current, producing either capillary obstructions or blood stasis in the parenchyma of the organs."

This account is entirely fanciful, and is evidently based upon erroneous observation and misinterpretation of what has been seen under the microscope, and upon imperfect methods of research. No such micro-organism as Freire has described is known to bacteriologists, and certainly nothing of the kind is to be found in the blood and tissues of yellow fever cases, as he asserts.

At the time of my visit to Brazil (1887), Dr. Freire, who had just returned from France, presented to me, as his yellow fever germ, a liquefying staphylococcus. Like other micro-organisms of the same class, this multiplies only by binary division; and having cultivated it in various media, I can say with confidence it does not form a black or a yellow pigment, and consequently does not correspond with the *Cryptococcus xanthogenicus* as previously described by Dr. Freire. Nor is it found in the blood and tissues of yellow fever patients.

Tetragenus febris flava.—The space allotted to this article will not permit me to give an extended account of other micro-organ-

isms which have been supposed to be concerned in the etiology of yellow fever. The *Tetragenus febris flava* of Dr. Finlay, of Havana, is a micrococcus in tetrads which I have frequently obtained from the surface of the body of persons not suffering from yellow fever in the city of Havana, and which I have now had in continuous cultivation for several years. It is not pathogenic; and it is not found, unless by rare exception, in the blood or tissues of yellow fever cadavers. The bacillus found by Dr. Paul Gibier in a certain number of cases of yellow fever studied by him, is only exceptionally present in the alvine discharges of yellow fever patients or in the contents of the intestine obtained as soon as possible after death. There is no satisfactory evidence that it bears any relation to the etiology of the disease.

Susceptibility.—Individuals of every race and of all ages, who are exposed to the yellow fever poison for the first time, during the epidemic prevalence of the disease, are liable to be attacked. But there is a wide difference in the degree of this susceptibility among races, and among individuals of the same race.

It has been asserted that the negro race has a congenital immunity from yellow fever; but this is a mistake. The susceptibility of the negro is, however, much less than that of the white race; and among those attacked the mortality, as a rule, is small.

This is shown by the statistics relating to the white and black troops in the British service at West India stations. "While in Jamaica the annual loss among the former amounts to 102 per 1000 of the mean strength, the deaths among the blacks did not exceed 8 per 1000. In the Bahamas the mortality of the whites was 59 in 1000, that of the blacks 5·6 in 1000" (La Roche).

In the Report of the Board of Experts appointed by Congress to investigate the epidemic of 1878, we find the following remarks:—

> Berwick City, 40 cases among coloured, no deaths. Morgan City, 21 deaths among coloured persons. Brownsville (Tenn.), of 162 coloured cases, 21 died. Chattanooga, of 685 cases, 256 whites, 429 coloured; of 164 deaths, 118 whites, 46 coloured. Decatur (Ala.), of 64 white cases, 28 died; of 168 coloured cases, 21 died.

The indigenous races of the West Indies and of the continents of North and South America have no immunity, except such as is acquired by residence in an endemic focus of the disease; and the same is true of the Mongolian race; but, like the negro, although in a minor degree, its susceptibility is less than that of the white race, and the mortality among those attacked is not so great.

In general it may be stated that the natives of northern latitudes

are more susceptible than those born in tropical or subtropical climates.

Blair, who had an extended experience in Guiana, says:—

> The lower the winter temperature in the native country of those attacked, the more severe was their sickness; so that, while the mortality among West Indians amounted to only 6·9 per cent. of the sick, it rose to 17·1 among the Italians and French, 19·3 among the English, 20·2 among the Germans and Dutch, and 27·7 among the Scandinavians and Russians.

Immunity is acquired by suffering an attack of the disease, or by long residence in localities where it is endemic, or prevails frequently as an epidemic; this acquired immunity is not, however, absolute. Second attacks no doubt occasionally occur, although this has been denied by some authors. Blair, whose experience was very great, says that he does not believe there is an instance of a second attack after a month's perfect restoration to health. Other authors are equally positive in their statements. On the other hand, we have numerous authentic accounts of second attacks.

Acclimatisation.—It is a remarkable fact that the population of a large city like Havana or Rio Janeiro, in which yellow fever has been endemic for a series of years, enjoys such a degree of immunity from the effects of the deadly poison that there is no interruption of business or pleasure at a time when strangers in the city are falling sick on every side. The development of an epidemic in these cities depends upon the presence of susceptible strangers in sufficient number to furnish a series of cases considered large enough to justify the use of the word. The presence of but few strangers during the epidemic season leads to the announcement that the disease is not epidemic, but that sporadic cases occur from time to time. Under exceptional circumstances, however, epidemics are developed in these endemic foci of the disease in which those who, by birth and long residence, were supposed to be acclimatised furnish a certain quota to the general mortality. This has frequently occurred, for example, in the city of New Orleans, where yellow fever formerly prevailed almost annually, and where the Creole population was supposed to enjoy an hereditary immunity.

HISTORY AND GEOGRAPHICAL DISTRIBUTION.

Restricted Range.—The geographical range of yellow fever is more restricted than that of any other acute infectious disease; and within the area of its prevalence it is, as we have seen, essentially a disease of the littoral, and especially of seaport cities.

While occasional epidemics have occurred upon the south-west coast of the Iberian peninsula, the disease, as an epidemic, is unknown elsewhere in Europe; and there is no evidence that it has ever invaded the great and populous continent of Asia. In Africa it is limited to the West Coast, and only to certain portions of that coast. In North America, although it has occasionally prevailed as an epidemic in every one of our seaport cities as far north as Boston, and in the Mississippi valley as far north as St. Louis, it has never established itself as an endemic disease within the limits of the United States. Vera Cruz, and probably other points on the Gulf Coast of Mexico, are, however, at the present time endemic foci of the disease. In South America it has prevailed as an epidemic at all of the seaports of the Gulf and Atlantic coasts, as far south as Monte Video and Buenos Ayres, and on the Pacific along the coast of Peru.

The region in which the disease has had the greatest and most frequent prevalence is bounded by the shores of the Gulf of Mexico, and includes the West India Islands. Within the past few years yellow fever has been carried to the west coast of North America, and has prevailed as an epidemic as far north as the Mexican port of Guaymas, on the Gulf of California.

Mode and Point of Origin.—The idea that yellow fever may originate *de novo* within the area of its occasional prevalence, was entertained by many medical authors during the first half of the present century, and still is held by a few. Thus Cornillac (1886) says: " In the zone which is habitual to it, yellow fever may develop at a given moment without apparent cause. It is born spontaneously at a point of this zone, or at several at a time; and neither the temperature, moisture, barometric pressure, electricity, or, finally, effluvia given off from the soil, can explain this sudden invasion." It is true that, in localities where the disease is endemic, cases occur which are not directly traceable to importation; but it is also true that in the principal endemic foci of the disease, such as Vera Cruz, Havana, and Rio Janeiro, yellow fever was at one time unknown; and we have reliable historical data fixing the date of its importation. In short, a careful consideration of the historical evidence relating to the disease gives no support to the idea of independent local origin, any more than in the case of smallpox, cholera, or other specific infectious diseases.

But the early history of the disease is involved in obscurity, and we are at present unable to determine whether, as maintained by some, it was endemic at certain points on the shores of the Gulf of Mexico at the time of the discovery of the " New World," or whether

it was imported to the West Indies from the African coast, as maintained by others.

Recent Extension to Brazil.—The highest medical authorities in Brazil agree that yellow fever was not endemic in the principal seaports of that republic prior to the year 1849, when it was introduced to the city of Bahia by the North American brig *Brazil*, which sailed from New Orleans, where yellow fever was prevailing, and touched at Havana. Two of the crew of this brig died of yellow fever during her voyage from the latter port to Bahia. Soon after her arrival the disease made its appearance among those who had communicated with the ship, and afterwards on other vessels in the harbour. The first case occurred a few days after the arrival of this brig (3rd November). A part of her cargo is said to have consisted of little barrels of beef which had become putrid. From Bahia the disease was carried to Rio Janeiro, where, during the epidemic season of 1850, it caused a mortality of 4160.

According to Professor Barata of the Faculty of Medicine of Rio Janeiro, yellow fever continued to prevail in Brazil until the year 1861, when it disappeared for eight years, to reappear in 1869–70, as the result of a fresh importation. The Italian ship *Creolla del Plata*, which had touched at St. Iago, where yellow fever was prevailing, is named as the vessel which introduced the disease on this occasion.

Epidemics in Rio de Janeiro.—The mortality from the disease under consideration in the city of Rio, from the time of its introduction in 1850 to a recent date, is shown by the following table:—

Year.	Mortality.	Year.	Mortality.	Year.	Mortality.	Year.	Mortality.
1850,	4160	1860,	1249	1869,	274	1878,	1174
1851,	475	1861,	247	1870,	1117	1879,	974
1852,	1943	1862,	12	1871,	8	1880,	1433
1853,	853	1863,	...	1872,	102	1881,	219
1854,	21	1864,	...	1873,	3659	1882,	95
1855,	...	1865,	...	1874,	829	1883,	1336
1856,	...	1866,	...	1875,	1292	1884,	618
1857,	1425	1867,	...	1876,	3317	1885,	278
1858,	800	1868,	...	1877,	282	1886,	1397
1859,	500						

Epidemics in the United States.—The mortality in the seaport cities of the United States which have suffered most severely from this exotic pestilential disease, as a result of favour-

able climatic conditions during the summer months, and of their proximity to permanently infected seaports in the West Indies, from the year 1800 to 1880, is shown by the following table:—

MORTALITY FROM YELLOW FEVER IN CHARLESTON (S. C.), PENSACOLA (FLA.), MOBILE (ALA.), NEW ORLEANS (LA.), AND GALVESTON (TEX.), DURING THE PRESENT CENTURY.

Year.	Charleston.	Pensacola.	Mobile.	New Orleans.	Galveston.
1800,	184	...	...	*	...
1801,	...	...	...	*	...
1802,	96	...	...	*	...
1803,	*	...	...	...	...
1804,	148	...	...	*	...
1805,	*	...	...	...	...
1806,	...	...	...	...	...
1807,	162	...	...	...	...
1808,	...	...	...	...	...
1809,	...	...	...	*	...
1810,	...	...	...	...	...
1811,	...	*	...	*	...
1812,	*	...	...	*	...
1813,	...	...	...	...	...
1814,	...	...	...	...	...
1815,	...	...	...	...	...
1816,	...	...	...	...	...
1817,	272	...	...	800	...
1818,	...	...	...	115	...
1819,	177	...	274	2190	...
1820,	...	...	...	*	...
1821,	...	...	*	...	...
1822,	2	257	*	239	...
1823,	...	...	...	1	...
1824,	235	...	*	108	...
1825,	2	*	*	49	...
1826,	...	...	...	5	...
1827,	64	*	*	109	...
1828,	26	...	*	130	...
1829,	...	...	130	215	...
1830,	30	...	...	117	...
1831,	...	...	...	2	...
1832,	...	...	...	18	...
1833,	...	...	...	210	...
1834,	49	*	...	95	...
1835,	25	...	...	284	...
1836,	...	...	...	5	...
1837,	...	...	350	442	...
1838,	351	...	...	17	...
1839,	134	*	650	452	250
1840,	22	...	...	3	...
1841,	*	*	*	594	...
1842,	...	*	60	211	...
1843,	1	*	240	487	...
1844,	...	*	*	148	400
1845,	...	*	...	2	...
1846,	...	*	...	160	...
1847,	...	*	76	2259	200
1848,	...	*	75	850	...
1849,	125	...	50	737	...
1850,	...	...	...	102	...
1851,	...	...	*	16	...
1852,	310	...	...	415	...
1853,	...	*	115	7970	536
1854,	627	*	*	2423	404
1855,	...	...	...	2670	...
1856,	211	...	...	74	...
1857,	13	...	...	199	...
1858,	717	*	*	3889	344
1859,	...	...	...	...	182
1860,	...	...	...	...	...
1861,	...	...	...	...	...
1862,	*	*	...	...	...
1863,	...	*	...	...	...
1864,	*	...	...	...	259
1865,	...	...	...	...	...
1866,	...	...	...	...	*
1867,	...	34	*	3093	1150
1868,	...	...	...	...	...
1869,	...	...	...	...	...
1870,	...	...	*	587	...
1871,	213	...	...	55	...
1872,	...	...	...	40	...
1873,	...	61	27	225	...
1874,	...	118	...	...	...
1875,	...	...	...	...	...
1876,	...	...	...	...	...
1877,	...	...	...	...	...
1878,	...	...	90	600	...
1879,	...	...	...	...	...
1880,	...	...	...	...	...

* Number of deaths not stated.

Since the year 1880 there has been but one epidemic within the limits of the United States. This occurred in the States of Florida and Alabama in the years 1887 and 1888, as a result of importa-

tion through the town of Tampa, a seaport on the Gulf Coast of Florida, which is in constant communication with Havana.

The exemption enjoyed for this considerable period (twelve years) by the cities of New Orleans, Mobile, Charleston, and other seaports, in which, as shown by our table, the disease formerly prevailed almost annually, is no doubt largely due to improved methods of quarantine administration.

Epidemics in Europe.—In Europe the ravages of yellow fever have been restricted mainly to the Iberian peninsula. This is due, no doubt, to the frequent intercourse between Spain and Portugal and the West Indian ports, in which the disease is most prevalent, and to the fact that the summer temperature of these countries is favourable for the epidemic extension of the disease; whereas the northern portions of Europe are practically outside of the yellow fever zone.

The first epidemic in Spain occurred in the year 1700 at Cadiz. This city also suffered in 1730–31, 1733–34, 1764, 1780, 1800, 1804, 1810, 1819–21. The epidemics of 1800, 1810, and 1819 were not limited to the city of Cadiz, but the disease extended to the interior, and caused a considerable loss of life in the provinces of Granada and Andalusia, and also in some of the towns of Murcia and Catalonia—especially in Barcelona, from which city the disease was conveyed to the island of Majorca during the last epidemic. No widespread epidemic has occurred in Spain since 1821; but local outbreaks, as a result of importation from the West Indies, have occurred at Gibraltar (1828), Barcelona (1870), and Madrid (1878).

The first epidemic at Lisbon was in 1723; a second was inaugurated in 1856, and during the following year developed into a devastating scourge, which extended to the towns of Belem, Olivaes, and Almada.

In Italy yellow fever has only once effected a temporary lodgment—at Leghorn in 1804, where it was imported from Spain.

Ships with yellow fever on board have occasionally arrived at English and French ports; but local conditions have apparently not been favourable to an extension of the disease, except to a limited extent at Brest in 1856, at St. Nazaire in 1861, and at Swansea (Wales) in 1864.

PATHOLOGICAL ANATOMY AND HISTOLOGY.

External Appearances of Body.—The exterior of the body of an individual who has recently succumbed to yellow fever presents an appearance which is quite characteristic. The

icteric colour of the skin, although often not noticeable during the last hours of life, is developed in a large majority of the cases very soon after death. The colour is that which is seen to follow a bruise in which there has been an effusion of blood, and the origin of the pigment is no doubt the same. The icterus from bile pigments, which occurs not infrequently during convalescence, and which may be seen in cases fatal from a relapse, or at a late period of the disease, gives a uniform saffron-yellow colour to the surface of the body and conjunctivæ. In the icteric discoloration of which we speak at present, the colour is not so intense and not so uniformly distributed. The depending portions of the body, and especially those subjected to pressure, have a deeper coloration, and are more or less livid and mottled from hypostatic congestion. Dutrouleau has shown that this ecchymotic appearance of the back is due to position and pressure by placing the body upon the abdomen or side. In this case it is still the most dependent part which shows the livid marbled appearance referred to. The face and hands frequently appear cyanosed, or the face may be livid and turgescent, like that of one recently drowned. A little stream of black vomit is frequently seen trickling down from the corners of the mouth, or a similar fluid may escape from the nostrils, and the lips and gums may be soiled with dark blood which has oozed from them.

Cadaveric rigidity is quickly established and well marked.

Internal Lesions and Pathological Changes.—*Brain.*—The appearances observed upon removing the calvarium are usually those of hyperæmia of the brain and its meninges. The pia mater is almost always congested, and the vessels of the brain are abnormally full of blood; the pons and medulla are especially in a condition of hyperæmia. There is more or less effusion into the sub-arachnoid space and in the ventricles; this is sometimes turbid, and has a yellow colour. The surface of the brain presents sometimes little hæmorrhagic points; and its substance, like the tissues of the body, generally has a more or less pronounced yellow tinge.

Blood Vessels and Capillaries.—Several of the most competent observers agree that there is a fatty degeneration of the walls of the small blood vessels and capillaries of the various organs, and account in this way for the hæmorrhagic tendency of the disease. This is certainly a more rational explanation than the one so frequently offered, viz. that the hæmorrhages are due to the disorganised and diffluent condition of the blood. A moment's reflection should show that this explanation is insufficient, and that the blood, however diffluent, cannot escape so long as the vessels are intact. Ecchymotic points, or hæmorrhagic infarctions, are

sometimes found upon the surface or in the substance of the heart, as well as in other muscles of the body and the organs generally. The cavities of the right side of the heart usually contain soft coagula or dark coloured fluid blood, and the right ventricle sometimes contains a more or less decolourised fibrinous clot.

Blood.—The blood in yellow fever has been described by many observers as "completely disorganised" as to its histological elements. This is a mistake, as is shown by the numerous photo-micrographs made by the writer while in Havana, in 1879, from the blood of cases near a fatal termination. Both the red and the white corpuscles retain their normal appearance, and I have frequently seen the leucocytes undergoing their characteristic movements, even after twenty-four hours, in blood which had been preserved in culture-cells. In the days of bleeding, numerous observers mentioned the fact that the blood either does not coagulate readily, or forms a soft, loose coagulum from which the serum does not separate. The same is true of the blood in the heart and large vessels after death; it is fluid and dark coloured. Although there is no general destruction of the red corpuscles, it is probable that a considerable number of these elements perish in severe cases, for the serum contains free hæmoglobin, which gives it a yellow colour even as early as the third or fourth day. This yellow colour is seen in the serum obtained from the application of blisters to the surface, and in blood drawn for microscopical examination. This passing of the hæmoglobin into the serum, which is no doubt the cause of the yellow discoloration of the tissues in yellow fever, has been ascribed to the presence of bile. This view is not sustained by the chemical researches of Cunisset, a recent French author, who has arrived at the following conclusions:—

"Yellow fever is not a poisoning by the bile; at the outset of the malady the biliary pigments are rarely found in the blood or in the urine. They appear generally only during the second period, and in a great number of cases they are not to be found at all.

"The biliary salts, of which the powerful action of 'deglobulation' admitted by certain authors might explain the disorders which the malady presents, do not exist in the matters vomited, or in the urine, or in the blood, except, in certain cases, in very feeble quantity. In view of the profound alterations of the liver, this absence of the biliary salts is easily understood, and the defective depuration of the blood is to be looked upon as a complication rather than a determining cause of the malady."

Stomach and Intestinal Canal.—The most important pathological changes in yellow fever are found in the organs contained in the

cavity of the abdomen. The mucous membrane of the stomach is always found to be more or less hyperæmic; the congestion is commonly not general, but is confined to smaller or larger spots or districts, in which it is observed to proceed from one or more centres. From these centres it extends or radiates in a lesser degree, either gradually to be lost, or to pass over to another congested district. It is owing to this peculiarity of the congestion that it presents no uniformity of character, but is observed to spread irregularly over larger or smaller portions of the membrane (Schmidt). In addition to these congested patches, there are often to be seen small red patches resembling ecchymoses, but which, according to Schmidt, consist of "an unbroken network of minute vessels congested with blood, and identical with that network of large capillaries which surrounds the aperture of the gastric glands." Crevaux believes that a fatty degeneration of the cells which line the gastric glands and the capillaries of the mucous membrane is the most important lesion here. Other authors speak of an acute gastric catarrh as the process indicated by the appearance and histological examination of the mucous membrane; others again deny that there is any inflammation. My own examination of thin sections stained in various ways, shows that in a certain proportion of the cases there is evidence of inflammation, as shown by the presence of an unusual number of leucocytes in the submucous coat. The dark fluid so often ejected during the last hours of life, known as black vomit, is almost always present in the stomach, in greater or less amount, after death. There is no question that the dark colour is due to the presence of blood pigment, more or less changed by the acid secretions of the stomach.

The present writer has repeatedly verified the fact that black vomit contains numerous decolourised blood corpuscles, in little agglomerations, surrounded by granules of pigment, which under the microscope is of a deep orange colour, rather than black.

The small intestine commonly contains more or less black matter, either fluid and resembling that found in the stomach, or mixed with mucous and smeared over the mucous coating, especially of the ileum. This, no doubt, comes partly from the stomach, but in other cases is due to passive hæmorrhage from the mucous membrane of the intestine itself. This membrane presents arborescent patches of congestion, or portions of the canal may be uniformly red from hyperæmia of the mucous coat; the colour varies from a pale red to a reddish-brown, and is usually more marked in the lower portion of the ileum than elsewhere. The large intestine occasionally

presents similar arborescent patches of congestion, but usually it has a normal appearance.

Liver.—The appearance of the liver in yellow fever is characteristic, at least so far as acute febrile diseases are concerned. Usually it contains less blood than in the normal condition, and is of a pale yellow or brownish-yellow colour, similar to that of new leather in its various shades; occasionally it is gorged with blood, and livid, deep blue, or dark purple in colour. In the victims of chronic alcoholism, it often presents the nutmeg appearance of cirrhosis. The dimensions do not differ materially from the normal, but the consistence is modified by the fatty change, which gives the characteristic colour, and the parenchyma is easily torn and more or less friable. On section it is found to be drier than in the normal state, except in the comparatively few cases in which it is hyperæmic; these are, as a rule, cases which have proved fatal at a very early period. A fact which indicates that there is a stage of congestion antedating that of anæmia and fatty degeneration. According to Crevaux, this congestion is located especially in the portal radicles surrounding the lobules, and is attended with œdema of the interlobular connective tissue. Whether this primary congestion is a constant phenomenon or not, it is certain that in a majority of the autopsies the liver is found to be anæmic, and to present more or less evidence of fatty change in the hepatic cells. This is not, however, a uniform process; but areas of greater or less extent are seen, in which the cells are infiltrated with fat globules, while in other places the cells appear normal. The fatty cells contain one or several fat globules of varying dimensions, and the protoplasm is reduced in quantity according to the extent of this fatty change. The nuclei very often remain intact in the cells infiltrated with fat; but, according to Schmidt, "a great number of the nuclei also undergo fatty degeneration." Often a collar of normal cells remains about the central vein, while those cells nearer the periphery of the lobule contain numerous fat globules.

Kidneys.—The kidneys are also the seat of important pathological changes in the disease under consideration. This consists essentially of a parenchymatous nephritis. Externally no material change is noted in the organs. They do not vary greatly from the normal size, and usually are normal in appearance. When the attack has been brief, however, they may be hyperæmic and of a deep red colour. Ecchymosed spots and hæmorrhagic foci are frequently seen beneath the capsule or in the cortical substance. In the latter situation little globular hæmorrhagic points, the size of a pin's head, have been observed, which, upon examination, proved to be the distended

capsules of the glomeruli. The change in the renal epithelium consists in a cloudy swelling, followed by fatty degeneration and desquamation. Every grade of change may be seen in the section, from a slight degree of cloudy swelling to complete disorganisation and desquamation of the cells. Whole bundles of tubes may often be seen which have been stripped of their epithelium and are entirely empty. In thin sections the lumen of the tubules is seen in places to be filled with infarctions of various appearance. Some are homogeneous and translucent, and it may be more or less coloured with blood pigment; these are composed of an albuminous material. Other infarctions are formed of the granular débris of the renal epithelium; or we may have a mixture of the granular and albuminous material, in which case the latter forms a matrix in which the granules are embedded. These infarctions correspond with the casts found in the urine; those of a granular character are most abundant, and in them the granules often have a fatty appearance. There are also accumulations which differ from these in the fact that they are deeply stained by the aniline colours. They seem to be made up of the nuclei of the cells, sometimes intact, although swollen and compressed; more commonly massed together, or broken into irregular fragments. At least this seems to me to be the most probable interpretation of those infarctions which are stained by nuclear staining agents.

PROPHYLAXIS AND INCUBATION.

General Prophylactic Measures.—What has been said as to the etiology of yellow fever indicates clearly enough the measures of prophylaxis to be taken in localities subject to invasion. These are—(*a*) exclusion of the exotic germ of the disease by the sanitary supervision, at the port of departure, of ships sailing from infected ports; and their thorough disinfection at the ports of arrival, when there is evidence justifying a reasonable suspicion that they are infected; (*b*) isolation of the sick on shipboard, at quarantine stations, and, so far as practicable, in recently infected places; (*c*) disinfection of excreta, and of the clothing and bedding used by the sick, and of localities into which cases have been introduced, or which have become infected in any way; (*d*) depopulation of infected places, *i.e.* the removal of all susceptible persons whose presence is not absolutely necessary for the care of the sick.

Of all measures of prophylaxis, those which relate to the sanitary improvement of cities and towns liable to become infected are perhaps the most important. Municipal hygiene has made great

strides since the early part of the present century, and it is probably to this fact, more than to any other, that certain northern cities, which formerly suffered severely from yellow fever epidemics, owe their long immunity from such visitations, *e.g.* New York and Philadelphia.

Individual Prophylaxis.—Individual prophylaxis requires the individual, first of all, to avoid infected localities. If it is absolutely necessary for a susceptible person to visit a place where yellow fever is prevailing, or to remain in one in which it has effected a lodgment, he should observe the following precautions: Keep away from low-lying and filthy portions of the city; avoid the vicinity of the wharves, and all localities known to be centres of infection, especially at night; sleep as far from the ground as possible; avoid excesses of all kinds, and especially in the use of alcoholic drinks; keep out of the sun during the hottest part of the day, and be careful not to become overheated by violent exercise; avoid constipation.

With reference to the method of prophylaxis by inoculation, practised in Brazil by Dr. Domingos Freire, and in Mexico by Dr. Carmona y Valle, the writer, after a careful examination, has reported officially that—

"There is no satisfactory evidence that the method of inoculation practised by Dr. Domingos Freire has any prophylactic value.

"The claims of Dr. Carmona y Valle, of Mexico, to have discovered the specific cause of yellow fever have, likewise, no scientific basis; and he has failed to demonstrate the protective value of his proposed method of prophylaxis."

Incubation.—The period of incubation in yellow fever does not usually exceed four or five days, and may be less than twenty-four hours. Instances of a much longer period of incubation have been given by various authors—even as long as six weeks or two months; but we are satisfied that these are due either to error in diagnosis, or to the fact that the cases resulted from the establishment of a new and unrecognised centre of infection. Those who believe that the disease is only communicated by personal contagion, naturally date the exposure to the latest date when such personal contact was possible. Thus, when a first case occurs on a ship at sea two or three weeks after leaving an infected port, the period of incubation is supposed to be at least this long. On the contrary, the attack is due, in all probability, to the fact that the ship is infected, and the first case will probably be quickly followed by others which have no direct connection with it, but result, as it did, from exposure on the infected vessel. Instances of an attack

occurring within twenty-four hours after arrival in a city where the disease was prevailing as an epidemic, are numerous and well authenticated.

It is proper to state that several authors, who have had great experience, believe that the period of incubation may be extended to fourteen, or even more days—Blair, Rush, Féraud.

Clinical History.

Onset and General Symptoms.—As a rule, an attack of yellow fever is not preceded by any well-marked premonitory symptoms. The attack may occur at night in one who went to bed in his usual state of health, or in the early morning after an uninterrupted sleep, or during the day while engaged in ordinary occupations. In other cases there is a feeling of lassitude and discomfort for two or three days prior to the attack, with loss of appetite, slight pain in the back and loins, a feeling of giddiness or slight headache, flatulent eructations, constipation, a tendency to perspire at night, or upon very slight exertion, and more or less muscular debility, together with a disinclination for any mental exertion.

The attack is commonly inaugurated with a more or less decided chill, which, by its violence and duration, affords some indication of the probable severity of the case. In certain grave forms of the disease the onset is insidious, and is not marked by any perceptible chill. In a considerable proportion of the mild cases also, especially in the tropics, there is no rigor, and the patient experiences at most only a slight sensation of coldness, which quickly gives place to that of heat. If a thermometer is placed in the axilla during the initial chill, it will be found that the temperature is already considerably above the normal, and very frequently it reaches the highest point attained during the entire attack within a few hours from its inception.

Accompanying the chill are other nervous phenomena, similar in kind to those attending the onset of other specific febrile diseases. There is cephalalgia, often very severe, and located by preference in the forehead and supraorbital region; the eyeballs also are painful, and there is intolerance of light in some instances. Pain in the loins is a very constant and early symptom, which occasions much distress, and sometimes extorts groans and cries from the patient. At the same time pain is usually experienced in the lower extremities, often of a very severe character, constituting the *coup de barre* of the French authors: it affects especially the calves of the legs, the

knees, and the ankles. These symptoms continue, and are even aggravated, after the rigor has passed and the febrile stage is fully developed; in the meantime the face becomes flushed, and sometimes deep red and swollen in appearance; the eyes are shining and suffused, the conjunctivæ more or less hyperæmic, and often deeply injected; in severe cases presenting a fiery, inflamed appearance, which is accompanied by photophobia; the skin becomes hot and dry, and there are apt to be, especially in patients of a nervous temperament, great restlessness and jactitation.

Course, Varieties, Special Symptoms.—*Temperature.*—Yellow fever is a disease of a single febrile paroxysm, lasting from forty-eight hours to seven or eight days—more commonly from three to five days. The acme of temperature is reached at the outset, and from this time the temperature line is a descending one, interrupted sometimes by a slight evening exacerbation, up to the termination of the first period of the disease—febrile stage. The second stage is characterised by great prostration of the vital powers, and lasts from a few hours to two or three days—stage of calm; the temperature during this stage sometimes remains a degree or more above the normal, but more commonly it is normal, or even subnormal for a time. This is followed in severe cases by a reactionary fever of irregular duration, which presents a more or less remittent character. These features are shown in the accompanying temperature charts (Fig. 36).

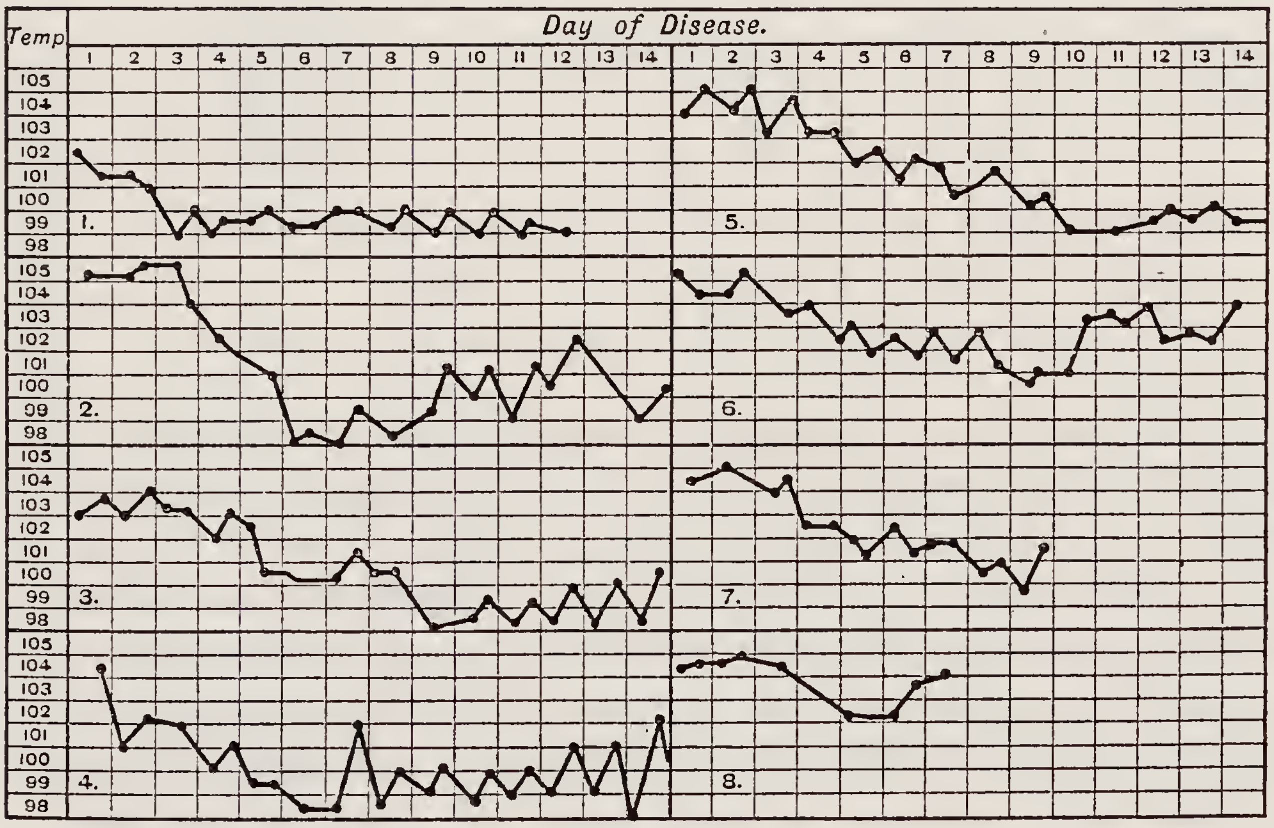

FIG. 36.

Naturally the typical temperature curve is disturbed by complications—visceral congestions, abscesses, parotitis, etc. It is also

disturbed by indiscretions in diet, over-active medication, and by moral causes, especially fright and grief. In mild cases the acme of temperature is reached during the first two or three hours of the attack. In more protracted and severe cases it is not reached until the second or third day, rarely later. In an analysis of 192 cases recorded by Faget, Jones, and myself, the acme was reached on the first day in 102, on the second in 54, on the third in 33, and on the fourth in 3. The highest temperature recorded by Faget was 107°·2 F. Thornton, in a total of 143 cases occurring at Memphis, noted a temperature of 108° F. in a single instance. With this exception, 105°·5 F. is the highest temperature recorded by him. In my own observations 106° F. has been the highest temperature noted. The temperature often rises rapidly just before death, and a very high post-mortem temperature (108° to 110° F.) is a common phenomenon. The temperature reached during the initial febrile paroxysm is a very good index of the severity of the attack, and furnishes a valuable basis for prognosis. This is shown by the following table, compiled by the writer some years since from a series of cases in which a complete and careful record had been made:—

Cases in which the Temperature was—	No. of Cases.	No. of Deaths.	Percentage of Deaths to Cases.
107° and above . .	13	13	100
106°–107° . . .	9	9	100
105°–106° . . .	36	22	61
104°–105° . . .	80	24	30
103°–104° . . .	87	6	7 nearly
102°–103° . . .	29	0	...
101°–102° . . .	15	0	...
Total, . .	269	74	27·5

Varieties of the Disease.—Systematic authors have described numerous varieties of the disease; but these are for the most part simply different grades in the degree of severity, or depend upon individual peculiarities and complications. Yellow fever is the same in all parts of the world where it occurs, and every great epidemic furnishes examples of the several varieties which have been described. It will suffice here to mention the classification adopted by one or two standard authors. La Roche gives an account of the clinical history of the disease under the following headings: *Inflammatory species*, including three grades, intense, mild, and ephemeral; *congestive species*, including four grades, aggravated, adynamic, walking, and apoplectic.

Béranger-Féraud, in his elaborate account of the disease as it prevails at Martinique, classifies the cases as follows:—

1*st Degree.*—Mild yellow fever.

2*nd Degree.*—Yellow fever of moderate intensity: (*a*) cases in which the onset is frank; (*b*) cases in which it is insidious.

3*rd Degree.*—Grave yellow fever: (*a*) ordinary forms, including the gastric, adynamic, ataxic, congestive, and typhoid forms; (*b*) rare forms, including the hyperæsthetic, gangrenous, algid or choleraic, and hydrophobic.

4*th Degree.*—Yellow fever *sidérante.*

Different epidemics are sometimes characterised by the predominance of one or the other of the forms described, and the character of the disease often varies greatly during the same epidemic. The earlier cases are sometimes mild and the mortality small, while later the greater intensity or malignancy of the poison is shown by the occurrence of a large number of cases of the severest grade, and even of those rapidly fatal attacks denominated by the French *sidérante.*

Pulse.—The pulse in sthenic cases is full, strong, and hard at the outset of the attack, and may reach 120 or more pulsations in the minute, more commonly not more than 100 to 110. It diminishes in rapidity and force as the disease progresses; and this occurs even when the febrile heat is not reduced for two or three days, and is considered by Faget a valuable diagnostic sign. During the second stage of the disease the pulse, however hard and accelerated it may previously have been, becomes preternaturally slow and soft. The feebleness of the heart, which seems to suffer especially from the action of the yellow fever poison, and which has undergone a certain degree of fatty degeneration, is shown by this very compressible and slow pulse, which is often reduced to 40, and sometimes even to 30 beats per minute. This constitutes a very characteristic feature of the disease, and affords an important indication for treatment during the period of depression or "calm."

Tongue.—The tongue is sometimes but slightly coated at the outset, and is usually moist; generally it quickly becomes covered with a white coating, which may be in streaks; the margins, as a rule, remain red. Very commonly the tongue is narrow and pointed, differing in this respect from the broad, flabby tongue of the malarial fevers. In the progress of the disease it often becomes dry, and the coating assumes a brownish colour, or it may become very foul, and loaded with sordes.

Face and Conjunctivæ.—The face is at first flushed or bright red and swollen, or it may be of a dusky violet hue; this, with the

deep red suffusion of the eyes in severe cases, is quite characteristic. The countenance often has an expression of anxiety or pain, or of dejection; again it may appear dull and indifferent.

In the last period of the disease, in fatal cases, the features become shrunken,—sometimes bloated and flabby,—the brows are often contracted, and the eyes sunken, with ecchymosed lids.

The hyperæmia of the conjunctivæ in mild cases may be temporary, in the more severe ones it is apt to last through the first period, and in quickly fatal cases the eyes may be deeply injected throughout. Usually by the third day a careful inspection will show that the conjunctivæ have a yellowish tinge, which becomes more intense as the disease progresses.

Skin.—The skin is hot and dry in some cases throughout the first period; in others it soon becomes moist, and there is a tendency to free perspiration, which is readily induced by covering with blankets, warm drinks, etc. Even in those cases in which the skin is hot and dry, the termination of the first period is marked by a soft, cool, and usually moist surface. In exceptional cases, the skin remains hot and dry up to the fatal termination. When death occurs in the stage of depression, the surface becomes cold, and is often covered with a clammy sweat. Many authors have spoken of a peculiar odour given off from the surface of yellow fever patients, and various attempts have been made to define its character by comparison with other known odours. Dr. Rush said that it resembled that of "the washings of a gun." Dr. Jackson describes it as "sickly and faint, and not unlike the smell of a fish-market."

The colour of the skin, which has given its name to the disease, is not always seen, but usually a yellow discoloration begins to make its appearance towards the end of the first period, and later becomes more intense, lasting for some time after convalescence is established. It varies much in intensity, from a slightly yellow tinge to a deep orange or saffron colour. In certain cases the skin presents a mahogany colour, or that of bronze. In fatal cases the yellow colour is developed immediately after death, even if it had not been very noticeable before dissolution took place.

The proportion of cases in which this yellow colour of the skin is observed varies greatly in different epidemics.

Various skin eruptions have been described as occurring occasionally, but there is nothing characteristic about any one of them unless it is the erythematous eruption about the scrotum, which Béranger-Féraud believes to be pathognomonic of the disease. Other eruptions mentioned as occurring occasionally are petechiæ, vesicular and pustular eruptions, livid spots and vibices, erythematous patches

about the knees and elbows, or a general erythematous eruption, papular eruptions, pustules about the mouth, furuncles, etc.

Urine.—The urine in yellow fever, even during the first period, is reduced in amount below the normal standard. This marked reduction, and in fatal cases very commonly complete suppression, is a notable feature of the disease.

The presence of albumen in greater or less amount is a symptom which is so constant that it has come to be generally accepted as one of the pathognomonic features of the disease.

The diminution in the amount of the urinary secretion in connection with the amount of albumen present is of importance in a prognostic sense, as it is to a certain extent an index of the gravity of the attack.

The most marked diminution occurs during the stage of depression, and the few ounces secreted in the twenty-four hours, in severe cases, are loaded with albumen to such an extent as to form coagula which occupy one-half to two-thirds of the contents of the test tube.

In mild cases only a slight trace of albumen may be found in the urine for a day or two, but usually the deposit is sufficiently abundant after the second day to leave no doubt as to its character. It becomes more abundant at the termination of the first stage, and in severe cases throughout the stage of calm, at which time suppression is very liable to occur, especially if, as a result of exposure, the cutaneous transpiration is checked. The early appearance and abundant presence of albumen is generally recognised as an evil prognostic. The reappearance of albumen, after it has once disappeared, indicates a relapse.

The amount of urea eliminated by the kidneys is less than normal, and in inverse proportion to the severity of the attack. According to Cunisset, the quantity is generally in proportion to the amount of urine secreted, and when the amount increases it is a favourable symptom. Uric acid is also said by this author to be present in diminished quantity, but in much less proportion than the urea. He says: "We have seen urine, containing only 7 grammes of urea per litre, give a relatively abundant deposit of uric acid." Bile pigments usually appear in the last days of sickness, and their presence is generally considered a favourable prognostic sign. In exceptional cases the urine may contain blood, from renal or vesical hæmorrhage. The urine in yellow fever almost invariably presents a decided acid reaction.

Nervous Symptoms.—The symptoms connected with the nervous system are varied. In mild cases the intellect remains undisturbed,

and only a moderate amount of transient pain in the head and back marks the onset of the attack. In severe cases the frontal headache and rachialgia are most distressing, and may last throughout the first period of the disease. This stage is also one of great restlessness and jactitation; the patient sleeps but little, and his sleep is apt to be disturbed by distressing dreams; the mind often seems to be in a state of tension; the patient is watchful, excited, and anxious. In other cases the mind is calm; and in others, again, there is a condition of apparent apathy or indifference. Delirium is not a very common symptom in yellow fever, and the intellect often remains unclouded throughout, even in fatal cases. Some cases, however, are attended with incoherency of ideas or hallucinations, and in some there is active delirium. More frequently the mind falls into a torpid condition, the patient is somnolent, and, when awakened, is disposed to be taciturn. A certain number of fatal cases are characterised by active delirium, and a greater number by coma gradually developed. In a limited number of cases death is preceded by convulsions, and occasionally tetanic symptoms of more or less general character have been noted. Among the nervous symptoms may be mentioned deep sighing respiration, sometimes of a spasmodic character. Naturally the respiration is increased in frequency during the febrile stage; in the stage of calm it again becomes normal in mild cases, or sighing and spasmodic in those of a graver character.

Symptoms referable to the Digestive System.—There is complete anorexia during the first stage of the disease; but when the remission occurs patients are very likely to desire food, and even to insist upon having it. Thirst is a constant symptom during the febrile stage, and also in the second period, especially when there is frequent and copious vomiting—black vomit. The bowels are commonly somewhat constipated at the outset. In the second period of the disease there is apt to be more or less diarrhœa, and in fatal cases a dark fluid is often discharged from the bowels, resembling precisely that from the stomach. Occasionally there is a discharge of pure blood, the result of intestinal hæmorrhage. A choleraic form of the disease is sometimes met with, in which there is profuse diarrhœa and collapse, similar to that in the algid stage of Asiatic cholera.

Symptoms connected with the stomach form a prominent part of the history of yellow fever. The characteristic black vomit which has so much occupied the attention of medical authors is for the vulgar the most striking feature of the disease; and even many physicians reserve their diagnosis in early cases during an epidemic

until they have had ocular evidence that the vomited matter is black. The common name of the disease in Spanish countries, "vomito," refers to this symptom. Yet in a majority of the cases there is no vomiting of black matter. Indeed, this is recognised as a very grave symptom; and although a considerable number of recoveries occur after the characteristic black vomit has been ejected, this may be considered an exception to the general rule.

Vomiting is a common symptom during the first period of the disease. Sometimes the fluid ejected has a yellow colour, from the presence of bile, but more frequently it is colourless, and consists only of the fluids ingested, containing, in suspension, a little mucus from the stomach; it is almost always acid. In favourable cases vomiting ceases with the first period of the disease; in those of a graver character, after an interval, perhaps, of twenty-four hours or more, during which there is more or less gastric distress, or a feeling of weight and discomfort, vomiting again occurs, either of a clear acid fluid, or of one of the varieties of black vomit. At first the black material may be in the form of little flocculi suspended in a transparent fluid, "coffee-ground vomit"; and as the case progresses the amount of this material increases, until the whole fluid appears to be uniformly black. Upon allowing it to stand, however, it commonly separates into two portions, and it will be seen that it still consists of a clear liquid with the black matter in suspension. Upon microscopic examination it will be found that the coloured material is made up of little masses which are not black, but have a yellowish-brown colour. A careful examination of recent specimens shows that these little masses contain decolourised and more or less deformed red blood corpuscles and granular leucocytes, and that the brown matter is diffused about these agglomerated cellular elements, and the matrix of mucus in which they often seem to be included. There is no doubt that the black vomit is due to passive hæmorrhage from the gastric mucous membrane, although this has been disputed by some recent authors—Freire, Carmona, Gibier. In certain cases there is vomiting of pure blood, resulting from an active gastric hæmorrhage. The black vomit is not always discharged during life, but it is extremely rare not to find it in the stomach after death.

The fact that the gastric mucous membrane is seriously involved in this disease, is shown not alone by the vomiting, the passive hæmorrhage, and the post-mortem appearances, but also by the constant feeling of discomfort or pain in severe cases, and by a marked tenderness upon pressure over the epigastrium.

Hæmorrhages.—Hæmorrhage may occur, not only from the mucous membrane of the stomach and intestine, but also from that of the

mouth, nose, bladder, and uterus, and even from the eyes or ears. In some epidemics epistaxis is quite common; it may occur during the first period of the disease, but it is more frequent during the second, at which time the general tendency to hæmorrhage is developed. Next to epistaxis, hæmorrhage from the buccal mucous membrane, and especially from the tongue, the gums, and the lips, is the most frequent.

Convalescence and Relapses.—In mild cases of yellow fever, convalescence is quickly established. Instead of the usual depression which marks the second stage of the disease, the patient, after a fever of three to five days' duration, may enter almost immediately into convalescence, and within a week may be ready to resume his usual avocations. But in severe cases, convalescence is often slow, and may be interrupted by various complications—parotitis, buboes, furuncles, abscesses, hepatitis, diarrhœa. In the army, experience shows that but few of those attacked are able to resume their duties in less than ten days, and a considerable number remain in hospital from thirty to fifty days. Blair found the average number of days during which patients were retained in hospital after convalescence was established to be 6·55 for mild cases, and 7·91 for grave cases.

Relapses occur for the most part during the early period of convalescence, or before it is fairly established; they are generally regarded as even more dangerous than the first attack. Occasionally a relapse, or second attack, occurs as late as from two to four weeks after the termination of the febrile period of the first attack.

PROGNOSIS AND MORTALITY.

Prognosis.—The prognosis in yellow fever should always be guarded; for not only is it a very fatal disease in its severer forms, but cases which appear mild at the outset may suddenly assume a grave character. It is more favourable in the case of women and children than with men, and is especially unfavourable in those of intemperate habits, and in plethoric persons who have recently arrived in the infected locality. The principal guide in forming a prognosis at the outset of a case is furnished by the temperature observations. When the body heat does not go above 103° to 103½° F. during the first two days, a favourable result may be anticipated. Later, the amount and character of the urinary secretion is the most important prognostic indication. When this is scanty and heavily loaded with albumen, the case is very grave, although the general appearance of the patient may be favourable,

and he may insist that he is "all right," and wants something to eat. On the other hand, hopes of a favourable issue may be entertained, even in cases characterised by great prostration, and in which the hæmorrhagic tendency is pronounced, if the urinary secretion is tolerably abundant, and the quantity of albumen small. The throwing up of black vomit is by no means a fatal sign; but, as a rule, the passive hæmorrhages which occur during the second stage of the disease are of grave import. Epistaxis occurring during the first stage has been considered by some authors a favourable event (?).

The early appearance of jaundice is an unfavourable sign; as are also frequent vomiting, and great distress and tenderness in the epigastric region. Intense and long continued injection of the conjunctivæ, giving the eyes a fiery red colour, is unfavourable. Delirium, great restlessness and jactitation, and sighing respiration, are all symptoms which give reason for anxiety as to the result.

Mortality.—The mortality from yellow fever varies greatly in different epidemics, and among different classes of the community.

Among the natives of the cities in which the disease is endemic, or in which it has frequently prevailed as an epidemic, it may be as low as from 7 to 10 per cent. One reason for this comparatively small mortality is found in the fact that a considerable proportion of the cases in such a community are among children. Among unacclimated adults the mortality ranges from 20 to 50 per cent., and under certain circumstances even exceeds the latter figure. In the great Philadelphia epidemics of the last and the beginning of the present century, the mortality was from 20 to 80 per cent. In the French Antilles, according to Dutrouleau, the mortality during the years 1851 to 1857, inclusive, ranged from 12·9 per cent. to 50 per cent. Blair gives the mortality at the Seamen's Hospital at Georgetown (Demerara) in 1838 at 19·5 per cent.

At Vera Cruz the mortality for seven years (1875–1881), according to Dr. Molina's statistics, was 41·78 per cent. in the hospital for men, and 41 per cent. in the hospital for women. In Rio Janeiro the mortality for the year 1870 was 17·4 per cent. In the epidemic of 1873 it was 23·3 per cent.

The mortality among the Spanish troops in Cuba, according to the statistics of Dr. Bastarreche, was, in the year 1855, 24·31 per cent., and in the royal navy 17·8 per cent.

Dr. Bemiss gives the mortality rate for the city of New Orleans, during the great epidemic of 1878, as 16·66 per cent.

TREATMENT.

Remedies that have been recommended.—The unsatisfactory results obtained from the various methods of treatment proposed, are shown by the fact that a majority of the physicians, in those parts of the world where yellow fever prevails, who have had an extended experience, agree that active medication is injurious, and have settled upon an expectant or symptomatic treatment, with careful nursing, as giving the most favourable results. Yet the records of the past show that a certain proportion of the cases recover under all modes of treatment, even the most active and opposed to our present views.

Emetics were formerly considered an essential part of the antiphlogistic treatment. We speak of them only to condemn them, except in those cases in which at the outset of an attack there is evidence that the stomach contains undigested and fermenting material which is the cause of nausea and distress. In this case a simple emetic may be administered, with the sole object of unloading the stomach.

Purgatives have at all times been considered useful at the outset of an attack of yellow fever; and it is the standard treatment, wherever the disease prevails, to administer a cathartic of some kind as soon as the patient comes under observation. Opinions differ as to the best form of cathartic medicine: some prefer a mercurial, some a saline, and others a full dose of oleum ricini. On account of its certain, prompt, and thorough action, and the absence of irritating properties, the last mentioned medicine is a favourite in our own southern cities, in the West Indies, and in Brazil, especially in domestic practice among the creole population. The dose usually given might seem excessive to those who have not seen its favourable action; half a tumblerful, or even more, is the common dose for an adult—2 to 4 fluid oz. Many physicians still prefer a mercurial purge, followed if necessary by a saline cathartic; and calomel is the mercurial usually selected.

It must be remembered that the gastro-intestinal mucous membrane is in a hyperæmic condition early in the attack; and experience has demonstrated that this condition is aggravated by active medication, or by the presence of food,—even the simplest,—and that the gravest danger may result from the administration of anything which may add to the irritation of the stomach or intestine, the normal functions of which seem to be arrested as the result of the action of the toxic agent which gives rise to the morbid phenomena. For this reason it is necessary to administer cathartics with great

caution, if they seem called for after the second day of the disease. As a rule, it will be best to spare the stomach, and to move the bowels with purgative enemata.

The prevailing opinion that yellow fever is closely allied to the malarial fevers, led many physicians, during the first half of the present century, to prescribe quinine in the expectation that it might prove a specific in this disease. The expectation was not fulfilled; but there are still a certain number of experienced physicians who think a full dose at the outset of an attack to be beneficial; others consider it injurious, and, as a rule, its use has been abandoned.

Blair, whose experience was very extensive, and whose treatment seems to have been unusually successful, gave 20 grains of calomel and 25 grains of quinine at the outset of an attack, and under certain circumstances repeated the dose several times.

Dr. Porcher, of Charleston, follows Blair's treatment so far as the first dose is concerned, but protests against a repetition of it.

Symptomatic Treatment.—*To reduce the Temperature.*—In the symptomatic treatment of the disease, sedative doses of aconite or of digitalis, or of one of the modern antipyretics—such as antipyrin—may be cautiously administered during the first stage; but it must be remembered that as the temperature falls and the patient passes into the second stage of the disease, the action of the heart is very feeble, and there is a tendency to death by syncope.

Baths of all kinds—hot, cold, tepid, medicated—have been given in yellow fever, and the evidence is varied as to their utility. The most judicious and experienced practitioners are, however, in accord as to the danger of disturbing the patient to the extent demanded by frequent baths, and, as a rule, content themselves with hot pediluvia at the outset of the attack, and with sponging the surface with cold water or evaporating lotions to reduce the temperature. In the southern part of our own country, and in the West Indies, a hot mustard foot-bath, administered at the outset of the attack, is a standard method of treatment. The patient is wrapped in a blanket, and sits with his feet and legs immersed in a bucket of water as hot as he can bear, to which a liberal quantity of mustard has been added. This has a tendency to relieve cerebral congestion and headache, and often produces a free perspiration. It may be repeated several times during the first twenty-four hours. Cold applications to the head, and repeated sponging of the hands, arms, and chest with cold water, give comfort to the patient, and are of decided benefit on account of their antipyretic effect. Some practitioners recommend the use of tepid water for sponging the surface;

Féraud prefers a mixture of one part of aromatic alcohol with three parts of water; Dr. Peyre Porcher recommends "the assiduous and protracted application of ice-cold water to the head, hands, and arms as long as they are abnormally hot."

Béranger-Féraud speaks highly of the use of large enemata of cold water, frequently repeated, as a means of reducing the temperature. He has made extensive use of this mode of treatment, and has never seen any evil results from it.

To relieve Visceral Congestion.—The use of sinapisms and vesicants to relieve visceral congestion by revulsion to the surface, is approved by experienced physicians everywhere. We prefer sinapisms, and believe that, as a rule, they will accomplish all that can be expected from a blister. The feeling of weight, distress, or absolute pain in the epigastric region, attended with nausea, which is largely due to hyperæmia of the gastric mucous membrane, is often notably relieved by the application of a sinapism to the epigastrium. The patient, who has been restless and uncomfortable, will sometimes quickly fall asleep after such an application. In the same way, cerebral congestion and headache may be relieved by revulsives to the extremities, and lumbar pain by the application of a large sinapism to the loins.

The scanty urinary secretion, and often complete suppression, in yellow fever, seems to call for the use of diuretics; but unfortunately experience teaches that the most reliable medicines of this class do but little good. The kidneys are best relieved by those means which promote perspiration, and by revulsants applied to the loins. Complete suppression is rarely, if ever, relieved by any plan of treatment. In cases with a hot, dry skin, and scanty urinary secretion, with lumbar pain, indicating congestion of the kidneys, we should expect benefit from the hypodermic administration of the muriate of pilocarpine.

To stimulate the Heart.—Stimulants are rarely required before the fourth or fifth day, and must be given at first cautiously and in small doses, so as not to disturb the stomach; later, however, they are often absolutely demanded to sustain the feeble heart. During the second stage of the disease, when the pulsations of the heart often do not exceed 40 or 50 to the minute, there is always a tendency to syncope. This is especially manifested during the night, or toward morning; and many a patient, whose condition seemed satisfactory at the evening visit, has been found by his physician dead the next morning. This dangerous period may often be tided over by the use of stimulants. When the stomach is very irritable, a little iced champagne will often answer better

than anything else; but perhaps the best form of stimulant is good brandy, given in teaspoonful doses at intervals of half an hour, more or less, according to circumstances. It should be given ice cold. Later, milk punch, English ale or porter, may be given in liberal quantities, especially to those who are in the habit of using spirits.

Blair, whose opinion in all that relates to this disease is worthy of attention, prefers hock to any other form of stimulant. He says: "Of all cordials the best is Rhenish wine. When of good quality, it is retained when everything else is rejected, and it is universally liked by the patients. I have seen the most excellent effects from its use, and have often given it to the extent of two bottles in twenty-four hours. I believe it has saved many lives, and know of no substitute for it."

To control Vomiting.—Medicines administered by the stomach to control vomiting, as a rule, are worse than useless. Minute doses of morphine, administered hypodermically, are sometimes useful in checking gastric irritability; but this is a dangerous remedy in yellow fever, and only very small doses are tolerated. Blair has seen stupor, prostration, and complete narcotism follow the administration of one-tenth of a grain. On account of the danger attending its administration, he thinks that it will be more judicious to place it in the *index expurgatorius* of yellow fever.

The Sternberg Treatment.—Recent experience during the last epidemic in the United States (1888) indicates that a mode of treatment suggested by the writer while pursuing his investigations in Cuba in 1887, has a very favourable influence in preventing or checking gastric irritability, and also in increasing the amount of the urinary secretion. The intensely acid condition of the urine and of the vomited matters, and the fact that I have usually found the contents of the intestine more or less acid, led me to think that a very decidedly alkaline treatment might be beneficial; and in view of the probability that the specific infectious agent is located in the alimentary canal, I combined with the antacid selected an antiseptic agent which is known to restrict the development of micro-organisms when present in very minute quantity.

The formula suggested was as follows:—

℞	Bicarbonate of soda, .	Grams x (150 grains).
	Bichloride of mercury, .	Centigrs. ii ($\frac{1}{3}$rd grain).
	Pure water, . .	Litre i (1 quart).

m Sig., 50 grams (3 tablespoonfuls) every hour; to be given ice cold.

Bichloride of mercury, in a comparatively small amount, was added to the formula, not with the idea that it would to any extent destroy micro-organisms in the intestine, but as an antiseptic, which might be useful in preventing fermentative changes in the stomach, which would perhaps be favoured by the free administration of an alkali. To what extent it is concerned in the favourable results which have been reported by several physicians who have employed this treatment, I am unable to say.

The mercuric chloride, which remains in solution in presence of sodium bicarbonate in the proportions prescribed, would be precipitated by potassium carbonate, which is also contra-indicated in a disease in which there is a great tendency to suppression of urine, and uræmic poisoning. The experiments of Zelz and Ritter, and of Bouchard, show that in uræmia the toxic symptoms are largely due to the retention of potassium salts rather than to urea.

The treatment referred to has been tested by a number of physicians in the United States, in Cuba, and in Brazil, and I have had reports of 374 cases treated by ten physicians. Of these, 301 were whites, with a mortality of 7·3 per cent.; and 73 cases blacks, with no mortality.

During the last epidemic in the United States, at Jacksonville, Florida, Dr. Sollace Mitchell treated 106 cases in the "Sand Hills Hospital" with 5 deaths, a mortality of 4·7 per cent. Of the 106 cases, 79 were whites, and of this number 73 were adult males. All of the deaths occurred among these; and the mortality among this class, considered separately, was 6·8 per cent.

The mortality among cases treated at the Mercedes Hospital, Havana, during a period of seven years (1882 to 1888), as shown by the official statistics, was 43·5 per cent. (total cases, 712). In 1889, Drs. La Guardia and Martinez treated 44 cases in this hospital by the method under consideration, with a mortality of 15·9 per cent. They say in reporting their results: "It appears that the Sternberg treatment has diminished the mortality to less than one-half the mean mortality of this hospital. We have observed the following facts: The patients have offered a notable gastric tolerance during the medication; when treated from the first day, vomiting has rarely occurred. The secretion of urine has always been considerable; even in grave cases, when deaths occurred, they did not die anuric."

Alimentation.—The alimentation in this disease, as in the specific fevers generally, is a matter of prime importance. During the first part of the febrile stage no food is desired, nor is it required. It is best to give nothing in the way of food for the

first three or four days of sickness; after this, if the stomach tolerates it, an ounce or two of iced milk, or of chicken broth, may be given every two or three hours. If the stomach is very irritable, a smaller quantity at shorter intervals may be given; and it will be best to give the milk in combination with lime water. If the stomach will not retain this, or if it gives distress, do not push it further, but support the strength by nutritive enemata.

Even when the stomach is quiet and the patient craves food, it will be necessary to give only liquid nourishment for two or three days, and then to allow nothing but the simplest and most digestible forms of solid food; for there is danger of a relapse from imprudence in diet, even in the mildest cases.

Illustrative Cases.

The following cases which occurred during the epidemic at Fort Barrancas, Florida, 1875, illustrate the clinical history and treatment of the disease:—

Case 1.

Private Rosenberg, aged 32, admitted to hospital at 12.30 A.M., July 21st. —Temperature, 105°; headache; pain in back and loins. To have ol. ricini ℥iss, and a hot mustard foot-bath. 7 A.M.—Temperature, 103°; tongue heavily coated; eyes natural. To have warm tea or cold water *ad lib.* 6 P.M.—Temperature, 104°; pulse, 70; respiration normal; skin perspiring; face flushed; eyes glistening; bowels moved twice. To have small pieces of ice, or iced water.

July 22nd.—Temperature, 104°; pulse, 64; eyes glistening; tongue coated in centre with red margins; in a profuse perspiration; bowels moved once. 6 P.M.—Temperature, 104°·5; urine during the day ℥ix, s. g. 1025, acid, non-albuminous.

23*rd.*—Morning temperature, 104°·5; feels better; no headache; pain still in back; urine ℥ix, s. g. 1020, acid, albuminous. 6 P.M.—Temperature, 103°; urine ℥ix, acid, s. g. 1020, albuminous; complains of pain in his stomach. Apply mustard poultice at 7.30 P.M. At 8. P.M. feels much relieved.

24*th.*—Temperature, 101°; slept some, and feels better; urine ℥viij, s. g. 1024, acid, albuminous. Continue iced water or pounded ice *ad lib.* 6 P.M. —Temperature, 102°; urine ℥xij, s. g. 1024, albuminous.

25*th*, 7 A.M.—Temperature, 100°; urine ℥xv, s. g. 1023, acid, non-albuminous. To have chicken broth, ℥i, every hour, alternately with whisky toddy, ℥ss. Evening temperature, 102°; pulse, 50.

26*th.*—Morning temperature, 99°·8; pulse, 46; urine ℥viij, s. g. 1020, non-albuminous. To have iced milk and brandy toddy. Evening temperature, 100°; pulse, 52; perspiring freely all day.

27*th.*—Morning temperature, 98°·4; pulse, 46. To have iced milk and milk toast; continue brandy toddy. Evening temperature, 99°.

28*th.*—Morning temperature, 98°·4. Observations upon the temperature

and urine were taken until August 2nd; but as convalescence progressed favourably, it is unnecessary to detail them.

Note.—Case reported by Surgeon Sternberg, U.S.A.

Case 2.

Corporal Berger, aged 24 years. Taken sick at 9 A.M., July 21st. Temperature, 105°. To have ol. ricini ℥iss, and a hot mustard foot-bath; warm tea or iced water, as preferred, *ad lib.* 6 P.M.—Temperature, 105°; pulse, 110; tongue heavily coated; is perspiring freely; face flushed; eyes congested. ℞—Hyd. chlor. mite et quinæ sulph. aa grs. x, at once.

July 22nd, 7 A.M.—Temperature, 104°; tongue slightly coated; bowels moved twice; pulse, 80; eyes congested; urine ℥iv, s. g. 1020, acid, albuminous; feels but little better. 6 P.M.—Temperature, 105°. 9 P.M.—℞, Quinæ sulph. grs. xv; temperature at 10 P.M., 105°·5. 11 P.M.—Feels a little better.

23rd.—Temperature at 7 A.M., 105°·5; urine ℥viij, s. g. 1020, acid, albuminous; feels no better. To have iced water or pounded ice *ad lib.* 6 P.M.—Temperature, 105°·5; headache, and is very restless.

24th.—Morning temperature, 103°; urine ℥xiv, acid, s. g. 1020, albuminous (highly). Feels better; slept some; vomited two or three times. To have pounded ice *ad lib.* 6 P.M.—Temperature, 103°.

25th.—Morning temperature, 100°; urine ℥xx, s. g. 1020, acid, albuminous; slept pretty well, and feels better. To have chicken broth ℥i, alternately with whisky toddy ℥ss, every two hours. Evening temperature, 101°·8; skin moist; pulse, 70.

26th.—Morning temperature, 100°; pulse, 64. Continue chicken broth and brandy toddy. Evening temperature, 101°.

27th.—Temperature, 99; pulse, 60. To have iced milk or chicken broth and brandy toddy. Evening temperature, 100°·6.

28th.—Diet: Milk, broth, and toast. Convalescence progressed favourably, and notes were taken upon the temperature and urine until August 3rd, on which day boiled chicken was allowed for dinner.

Note.—Case reported by Surgeon Sternberg, U.S.A.

Case 3.

Private Spaulding, aged 26 years. Taken sick during the night of July 26th. No chill.

July 27th.—Morning temperature, 104°·5; pulse, 118; headache; eyes injected. ℞—Ol. ricini ℥iss, and hot mustard foot-bath. Evening temperature, 104°·4; urine ℥iv, s. g. 1035, acid, non-albuminous; tongue clean; perspiring freely; feels easy except slight headache.

28th.—Morning temperature, 103°·8; pulse, 94; bilious vomiting last night, but feels better this morning; no nausea; urine ℥iv, s. g. 1031, slightly albuminous. Evening temperature, 104°·8; pulse, 96.

29th.—Morning temperature, 104°; pulse, 96; urine ℥viij, s. g. 1025, slightly albuminous; perspiring freely; vomited a little last night. To have champagne. Evening temperature, 104°; pulse, 85; vomiting ceased; hæmorrhage from nose; urine ℥viij, s. g. 1025, albuminous.

30th.—Morning temperature, 102°; pulse, 90; urine ℥vj, s. g. 1025, slightly albuminous; bowels acting freely; slight hæmorrhage from the nose. To have chicken broth. Evening temperature, 103°·5; has been vomiting. To have iced champagne. Urine ℥vj, s. g. 1031.

31*st.*—Morning temperature, 102°; pulse, 84; urine ℥viij, s. g. 1020, albuminous; hæmorrhage from the nose last night. ℞—Ext. ergot fl. ʒi; beef-tea ℥i, every hour. Evening temperature, 101°·4; pulse, 78; urine ℥v, s. g. 1020, albuminous; nose bled to-day; feels very weak. To have champagne.

August 1*st.*—Morning temperature, 101°; pulse, 98; urine ℥viij, s. g. 1018, albuminous; hæmorrhage from the nose continues. Apply tr. ferri perchlor. Evening temperature, 102°; pulse, 80; gums bleeding. Chlorate of potass and iron (tr. ferri chlor.) mouth wash. Urine ℥xiv, s. g. 1015, albuminous.

2*nd.*—Morning temperature, 100°·5; urine ℥xviij, s. g. 1019, albuminous; bleeding from gums ceased. To have milk punch. Evening temperature, 101°·5; violent epistaxis. ℞—Ext. ergotæ fl. ʒi, every hour until relieved. Urine not collected.

3*rd.*—Morning temperature, 101°; urine ℥xviij, s. g. 1039, albuminous; bleeding from nose ceased for a while during night; commenced again on picking his nose. ℞—Acidi gallici ℈i, aquæ ℥i. ♏ A tablespoonful. Evening temperature, 100°·5; urine ℥xiv, s. g. 1015, albuminous. Gallic acid controlled epistaxis, but it was renewed on his picking his nose. Gallic acid to be repeated.

4*th.*—Morning temperature, 100°·5; urine ℥xxiv, s. g. 1013, albuminous; nose bled all night; very weak. Hypodermic injection of ergotine, grs. iii. To have milk punch. Evening temperature, 101°; urine ℥v, s. g. 1011, albuminous; pulse feeble; epistaxis ceased after hypodermic injection; very weak and pallid. Hiccough during the day; stopped by champagne.

5*th.*—Morning temperature, 102°·5; feels stronger this morning; pulse fuller; breathing laboured; no more hæmorrhage; urine ℥xiv, s. g. 1010, non-albuminous. To have milk punch and beef-tea. Evening temperature 100°; urine ℥vij, s. g. 1010, albuminous. Is very weak; sinking. Died at 1 A.M.

Case reported by A. A. Surgeon Salomon.

Remark.—A good example of a hæmorrhagic case in which death finally resulted from exhaustion. It is worthy of note that the epistaxis ceased after the hypodermic injection of 3 grs. of ergotine after all other means had failed to arrest it.

CASE 4.

Hospital Steward W. E. Hill, aged 26 years. Taken sick during the morning of July 28th; no chill; general feeling of malaise; took mass. hyd. grs. x, last night; had a hot mustard foot-bath this morning. Temperature, 100°·5; pulse, 86; face flushed.

July 29*th.*—Morning temperature, 100°·8; pulse, 72; urine ℥viij, s. g. 1028, non-albuminous; perspiring freely; feels cheerful; don't care for any nourishment. To have orange-leaf tea *ad lib.* Evening temperature, 100°; complained of pain in abdomen, relieved by application of flaxseed poultice; urine ℥vj, s. g. 1030, non-albuminous.

30*th.*—Morning temperature, 101°·5; pulse, 91; urine ℥viij, s. g. 1025, non-albuminous.

31*st.*—Morning temperature, 102°; pulse, 82; urine ℥vi, s. g. 1027, non-albuminous; slept well, but feels weak. To have chicken broth and arrow-root. Evening: Feels stronger; temperature, 101°·8; pulse, 90; urine ℥iv, s. g. 1025, non-albuminous.

August 1*st.*—Morning temperature, 101°·5; pulse, 90; urine ℥v, s. g.

1027, non-albuminous. Evening temperature, 101°; pulse, 78; urine ℥v, s. g. 1030.

2*nd.*—Morning temperature 101°; urine ℥iv, s. g. 1030, non-albuminous. Continue diet. Evening temperature, 99°·5; urine ℥vi, s. g. 1027, non albuminous.

3*rd.*—Morning temperature, 106°. Relapse, produced by exposure to cold during the night; the night was chilly, the thermometer having fallen to 65°, and the patient, having thrown off his bedclothes, was chilled. To have a hot mustard foot-bath, and orange-leaf tea *ad lib.* Urine ℥vij, s. g. 1028, non-albuminous. Evening temperature, 104°; feels better; perspiring freely; urine ℥v, s. g. 1025, non-albuminous.

4*th.*—Morning temperature, 104°·5; feels very well; skin moist; urine ℥vij, s. g. 1026, non-albuminous. Evening temperature, 106°. To have a hot foot-bath. No action of bowels. To have ol. ricini ℥i, ol. terebinth, ℥ss, per enema. Urine ℥vi, s. g. 1023, non-albuminous.

5*th.*—Morning temperature, 104°·2; urine ℥xij, s. g. 1025, non-albuminous; feels better; is perspiring freely. Evening temperature, 105°; urine ℥vj, s. g. 1020, non-albuminous.

6*th.*—Morning temperature, 103°. ℞—Potass. bromidi ʒss, every hour. Urine ℥x, s. g. 1017, albuminous. Evening temperature, 102°·6; urine ℥viij, s. g. 1018, albuminous. Has taken six doses of bromide; very drowsy.

7*th.*—Morning temperature, 101°·5; urine ℥xviij, s. g. 1014, albuminous. Very weak. To have champagne freely and beef tea. Evening temperature, 102°·5; urine ℥xj, s. g. 1015, highly albuminous. Is a little stronger; pulse fallen. Champagne does not agree, has vomited twice; ale to be substituted. Continue chicken broth.

8*th.*—Very listless, and inclined to sleep all the time. Morning temperature, 101°·8; urine ℥v, s. g. 1010, albuminous. Evening temperature, 104°; still inclined to sleep, and mind wanders. ℞—Liq. ammon. acetat. ℥ss, every hour. 9 P.M.—Took a sudden change for the worse; pulse hardly perceptible; sinking fast; died at 9.45 P.M.

Case reported by A. A. Surgeon Salomon, under direction of Assistant Surgeon Harvey E. Brown, U.S.A.

Remarks.—This is a characteristic case of fatal relapse, occurring as a result of improper exposure in a case which at the outset was of the mildest character.